

WEST Search History

DATE: Monday, February 28, 2005

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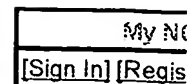
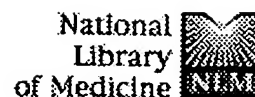
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END OF SEARCH HISTORY



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Items 1 - 195 of 195


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- ☐ 1: [Rosell R, Taron M, Sanchez JM, Moran T, Reguart N, Besse B, Isla D, Massuti B, Alberola V, Sanchez JJ](#) Related Articles, Links
 The promise of pharmacogenomics: gemcitabine and pemetrexed. Oncology (Huntingt). 2004 Nov;18(13 Suppl 8):70-6. Review. PMID: 15655942 [PubMed - indexed for MEDLINE]
- ☐ 2: [Wang YC, Lin RK, Tan YH, Chen JT, Chen CY, Wang YC](#) Related Articles, Links
 Wild-type p53 overexpression and its correlation with MDM2 and p14ARF alterations: an alternative pathway to non-small-cell lung cancer. J Clin Oncol. 2005 Jan 1;23(1):154-64. PMID: 15625370 [PubMed - indexed for MEDLINE]
- ☐ 3: [Nanda A, Buckhaults P, Seaman S, Agrawal N, Boutin P, Shankara S, Nacht M, Teicher B, Stampfl J, Singh S, Vogelstein B, Kinzler KW, St Croix B](#) Related Articles, Links
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- ☐ 4: [Liu Y, Chen Q, Zhang JT](#) Related Articles, Links
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- ☐ 6: [Gomyo Y, Sasaki J, Branch C, Roth JA, Mukhopadhyay T](#) Related Articles, Links
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-  **8:** [Fujimori S, Abe Y, Nishi M, Hamamoto A, Inoue Y, Ohnishi Y, Nishime C, Matsumoto H, Yamazaki H, Kijima H, Ueyama Y, Inoue H, Nakamura M.](#) [Related Articles, Links](#)


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-  **10:** [Chang YS, Wang L, Suh YA, Mao L, Karpen SJ, Khuri FR, Hong WK, Lee HY.](#) [Related Articles, Links](#)


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



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
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HER2-targeted therapy reduces incidence and progression of midlife mammary tumors in female murine mammary tumor virus huHER2-transgenic mice.
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Skp2 gene copy number aberrations are common in non-small cell lung carcinoma, and its overexpression in tumors with ras mutation is a poor prognostic marker.
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Molecular and immunological evaluation of the transcription factor SOX-4 as a lung tumor vaccine antigen.
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Overexpression of E2F-1 in lung and liver metastases of human colon cancer is associated with gene amplification.
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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


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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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
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










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
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
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
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



















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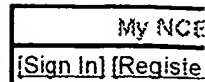
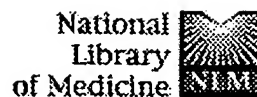
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Aneuploidy and cancer.

Sen S.

University of Texas, M.D. Anderson Cancer Center, Department of Laboratory Medicine, Houston 77030, USA. ssen@mdanderson.org

Numeric aberrations in chromosomes, referred to as aneuploidy, is commonly observed in human cancer. Whether aneuploidy is a cause or consequence of cancer has long been debated. Three lines of evidence now make a compelling case for aneuploidy being a discrete chromosome mutation event that contributes to malignant transformation and progression process. First, precise assay of chromosome aneuploidy in several primary tumors with in situ hybridization and comparative genomic hybridization techniques have revealed that specific chromosome aneusomies correlate with distinct tumor phenotypes. Second, aneuploid tumor cell lines and in vitro transformed rodent cells have been reported to display an elevated rate of chromosome instability, thereby indicating that aneuploidy is a dynamic chromosome mutation event associated with transformation of cells. Third, and most important, a number of mitotic genes regulating chromosome segregation have been found mutated in human cancer cells, implicating such mutations in induction of aneuploidy in tumors. Some of these gene mutations, possibly allowing unequal segregations of chromosomes, also cause tumorigenic transformation of cells in vitro. In this review, the recent publications investigating aneuploidy in human cancers, rate of chromosome instability in aneuploidy tumor cells, and genes implicated in regulating chromosome segregation found mutated in cancer cells are discussed.

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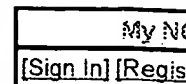
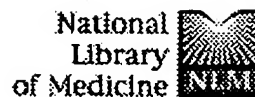
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 Comput Biol Chem. 2004 Dec;28(5-6):409-16.
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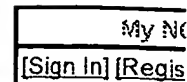
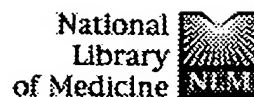
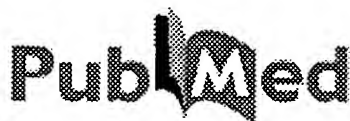
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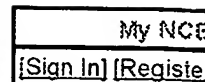
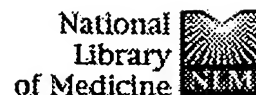
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Analysis of genomic and proteomic data using advanced literature mining.

Hu Y, Hines LM, Weng H, Zuo D, Rivera M, Richardson A, LaBaer J.

Institute of Proteomics, Harvard Medical School-BCMP, 240 Longwood Avenue, Boston, Massachusetts 02115, USA.

High-throughput technologies, such as proteomic screening and DNA microarrays, produce vast amounts of data requiring comprehensive analytical methods to decipher the biologically relevant results. One approach would be to manually search the biomedical literature; however, this would be an arduous task. We developed an automated literature-mining tool, termed MedGene, which comprehensively summarizes and estimates the relative strengths of all human gene-disease relationships in Medline. Using MedGene, we analyzed a novel micro-array expression dataset comparing breast cancer and normal breast tissue in the context of existing knowledge. We found no correlation between the strength of the literature association and the magnitude of the difference in expression level when considering changes as high as 5-fold; however, a significant correlation was observed ($r = 0.41$; $p = 0.05$) among genes showing an expression difference of 10-fold or more. Interestingly, this only held true for estrogen receptor (ER) positive tumors, not ER negative. MedGene identified a set of relatively understudied, yet highly expressed genes in ER negative tumors worthy of further examination.

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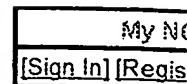
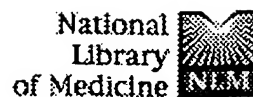
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[Using the method of two-color fluorescence in situ hybridization to detect aneuploidy of human sperms] Zhonghua Yi Xue Yi Chuan Xue Za Zhi. 1999 Apr;16(2):116-8. Chinese. PMID: 10194261 [PubMed - indexed for MEDLINE]

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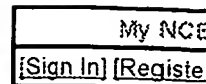
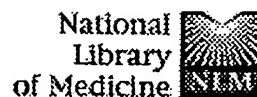
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Sen S.

University of Texas, M.D. Anderson Cancer Center, Department of Laboratory Medicine, Houston 77030, USA. ssen@mdanderson.org

Numeric aberrations in chromosomes, referred to as aneuploidy, is commonly observed in human cancer. Whether aneuploidy is a cause or consequence of cancer has long been debated. Three lines of evidence now make a compelling case for aneuploidy being a discrete chromosome mutation event that contributes to malignant transformation and progression process. First, precise assay of chromosome aneuploidy in several primary tumors with in situ hybridization and comparative genomic hybridization techniques have revealed that specific chromosome aneusomies correlate with distinct tumor phenotypes. Second, aneuploid tumor cell lines and in vitro transformed rodent cells have been reported to display an elevated rate of chromosome instability, thereby indicating that aneuploidy is a dynamic chromosome mutation event associated with transformation of cells. Third, and most important, a number of mitotic genes regulating chromosome segregation have been found mutated in human cancer cells, implicating such mutations in induction of aneuploidy in tumors. Some of these gene mutations, possibly allowing unequal segregations of chromosomes, also cause tumorigenic transformation of cells in vitro. In this review, the recent publications investigating aneuploidy in human cancers, rate of chromosome instability in aneuploidy tumor cells, and genes implicated in regulating chromosome segregation found mutated in cancer cells are discussed.

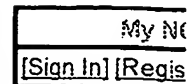
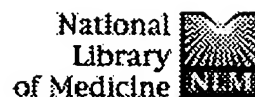
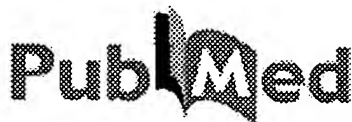
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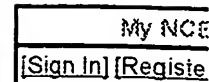
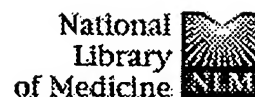
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Proteome analysis: biological assay or data archive?

Haynes PA, Gygi SP, Figeys D, Aebersold R.

Department of Molecular Biotechnology, University of Washington, Seattle 98195, USA.

In this review we examine the current state of proteome analysis. There are three main issues discussed: why it is necessary to study proteomes; how proteomes can be analyzed with current technology; and how proteome analysis can be used to enhance biological research. We conclude that proteome analysis is an essential tool in the understanding of regulated biological systems. Current technology, while still mostly limited to the more abundant proteins, enables the use of proteome analysis both to establish databases of proteins present, and to perform biological assays involving measurement of multiple variables. We believe that the utility of proteome analysis in future biological research will continue to be enhanced by further improvements in analytical technology.

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